EXHIBIT A

Expert Report of Lisa A. Bailey, Ph.D.

In the Case of:
Richard Sparks *v*. United States

Prepared by

Lisa A. Bailey, Ph.D.

Prepared for
United States Department of Justice
950 Pennsylvania Avenue NW
Washington DC 20530

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GRADIENT
www.gradientcorp.com
One Beacon Street, 17th Floor
Boston, MA 02108
617-395-5000

Table of Contents

List of Tables

4 Brief History of the US Marine Corps Base Camp Lejeune Site

4.1 Site Description and History

In the early 1940s, the United States Marine Corps developed a water-distribution system at its Camp Lejeune base, which is located in Onslow County, North Carolina, approximately 70 miles northeast of Wilmington, North Carolina (ATSDR, 2013a). The sole source of drinking water at Camp Lejeune is groundwater wells that pump water from the Castle Hayne aquifer systems (ATSDR, 2013a).

Operations at Camp Lejeune started in late 1941. Multiple water treatment plants (WTPs)[2] have serviced Camp Lejeune, including Hadnot Point (HP), Tarawa Terrace (TT), and Holcomb Boulevard (HB) (the three at issue in this litigation). The HP WTP was the first plant to come online (in 1942) and serviced the base until the TT and HB WTPs came online in 1952 and in the summer of 1972, respectively (Hennet, 2024). Because the WTPs were connected to many more groundwater wells than were needed to supply drinking water to the base, the wells' service was rotated and water from different wells was sometimes mixed at the WTPs before being delivered to Camp Lejeune residences and facilities as tap water (ATSDR, 2013a).

4.2 Investigations of Groundwater Contamination

In 1974, the Safe Drinking Water Act (SDWA) was established to protect the quality of drinking water in the United States (US Congress, 1974). Under the SDWA, US EPA developed national drinking water regulations that included the derivation of maximum contaminant levels (MCLs), *i.e.*, the highest level of a contaminant that is allowed in drinking water.

In the early 1980s, the groundwater sources for two of the WTPs that serviced Camp Lejeune (HP and TT) were found to be contaminated with volatile organic compounds. Although the groundwater source for the HB WTP was not contaminated, the HB WTP was contaminated when HB drinking water was supplied by the HP WTP in the spring and summer months from 1972 through 1985 (ATSDR, 2017a). The contaminants identified in the drinking water at the HP WTP were TCE, PCE, vinyl chloride, and refined petroleum products (including benzene) (ATSDR, 2017a). The HP contamination is believed to have been related to historical base operations and disposal practices (ATSDR, 2017a). TCE was the primary contaminant identified at the HP WTP. Groundwater modeling conducted by ATSDR estimated that the maximum mean monthly reconstructed level of TCE was 783 parts per billion (ppb), in November 1983 (ATSDR, 2017a). The maximum reconstructed mean monthly concentrations of benzene and PCE were 12 ppb (in April 1984) and 39 ppb (in November 1983), respectively (ATSDR, 2017a). The maximum reconstructed mean monthly concentration of vinyl chloride was 67 ppb, in November 1983 (Maslia *et al.*,

[2] Hadnot Point (HP), Tarawa Terrace (TT), and Holcomb Boulevard (HB) supplied drinking water to residences and workplaces at Camp Lejeune (see Hennet [2024]). Additional Camp Lejeune water-distribution systems which were not contaminated include: Marine Corps Air Station New River, Onslow Beach, Courthouse Bay, Camp Geiger, Rifle Range, and Montford Point/Camp Johnson (Hennet, 2024).

2016; ATSDR, 2017a). The maximum reconstructed mean monthly concentration of 1,2-tDCE was 435 ppb, in November 1983 (ATSDR, 2017a).[3]

Contamination of the TT WTP supply wells was found to be due to an off-site dry cleaner (Bove *et al*., 2014), with PCE identified as the primary contaminant. TCE, vinyl chloride, and 1,2-tDCE were also detected at this WTP as PCE degradation products (ATSDR, 2017a; Bove *et al*., 2014).[4] Groundwater modeling conducted by ATSDR, including a multispecies degradation model of PCE, estimated that the maximum reconstructed mean monthly concentration of PCE in the TT WTP was 158 ppb, in June 1984 (ATSDR, 2017a). Applying the same model, ATSDR estimated maximum reconstructed mean monthly concentrations of TCE and vinyl chloride of 7 and 12 ppb, respectively (ATSDR, 2017a). The maximum reconstructed mean monthly concentration of 1,2-tDCE was 22 ppb (ATSDR, 2017a).[5]

The wells directly serving the other Camp Lejeune water-distribution systems – Holcomb Boulevard (HB), Marine Corps Air Station New River, Onslow Beach, Courthouse Bay, Camp Geiger, Rifle Range, and Montford Point/Camp Johnson – were not contaminated with solvents (Hennet, 2024). As stated previously, the HB WTP was largely uncontaminated except when HB drinking water was supplied by the HP WTP (ATSDR, 2017a).

By February 1985, the most highly contaminated wells servicing the HP and TT WTPs had been removed from service (ATSDR, 2017b).

[3] Drs. Hennet and Spiliotopoulos explain in their expert reports that ATSDR's modeled groundwater concentrations are unreliable and likely biased high as a result of several conservative assumptions used in ATSDR's modeling (Hennet, 2024; Spiliotopoulos, 2024).

[4] Refined petroleum products were not contaminants of the TT WTP; therefore, benzene was not identified as a contaminant of concern at the TT WTP, and ATSDR did not model groundwater concentrations for benzene for the TT WTP (ATSDR, 2013b; Hennet, 2024).

[5] Drs. Hennet and Spiliotopoulos explain in their expert reports that ATSDR's modeled groundwater concentrations are unreliable and likely biased high as a result of several conservative assumptions used in ATSDR's modeling (Hennet, 2024; Spiliotopoulos, 2024).

5.2 Toxicity Criteria

This section summarizes the non-cancer toxicity criteria that US EPA derived for TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE based on the methodology described in Section 3, and US EPA's hazard assessment of these chemicals as described in the documents cited below. As described in Section 3, non-cancer toxicity criteria are derived based on the most sensitive endpoint identified by the regulatory agency deriving the value; none of these toxicity criteria are directly relevant to PD. In its PHA for Camp Lejeune, ATSDR also derived non-cancer TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE toxicity criteria for several health effects other than the most sensitive, including for neurological effects (ATSDR, 2017b); these values are also described below.

5.2.1 Trichloroethylene (TCE)

5.2.1.1 US EPA Toxicity Criteria (RfDs and RfCs)

Table 5.1 summarizes the points of departure (PODs), total uncertainty factors (UFs), candidate RfDs and effects associated with each, and the final RfD US EPA derived for TCE (US EPA, 2011b). Table 5.2 summarizes the PODs, UFs, candidate RfCs and effects associated with each, and the final RfC US EPA derived for TCE (US EPA, 2011b).

Based on the hazard assessment for TCE, US EPA derived three candidate RfDs from three rodent TCE drinking water studies (US EPA, 2011b); one based on fetal heart malformations in rats (Johnson *et al.*, 2003); one based on altered immune system endpoints in mice (Peden-Adams *et al.*, 2006); and one based on decreased thymus weight in mice (Keil *et al.*, 2009). US EPA used the average of these candidate RfDs as the final RfD (Table 5.1). US EPA then applied a TCE physiologically based pharmacokinetic (PBPK) model to conduct a route-to-route (oral-to-inhalation) extrapolation to derive the TCE RfCs from two studies used for the TCE RfD derivations (US EPA, 2011b) (Table 5.2).

Table 5.1 US EPA TCE Non-cancer Chronic Oral Toxicity Values

Chemical	POD (mg/kg-day)	UF	Candidate RfD (mg/kg-day)	RfD (mg/kg-day)	Effect/Source
TCE	0.0051	10[a]	0.00051	0.0005	Fetal cardiac abnormalities in rats (Johnson *et al.*, 2003)
	0.37	1,000[b]	0.00037		Altered immune system in mice (Peden-Adams *et al.*, 2006)
	0.048	100[c]	0.00048		Decreased thymus weight in adult female mice (Keil *et al.*, 2009)

Notes:
LOAEL = Lowest Observed Adverse Effect Level; mg/kg-day = Milligram per Kilogram Body Weight per Day; POD = Point of Departure; RfD = Reference Dose; TCE = Trichloroethylene; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: US EPA (2011a,b).
(a) A UF_A of 3 was applied to account for interspecies toxicodynamic differences, and a UF of 3 was applied to account for differences in sensitive populations, for a total UF of 10.
(b) A UF_L of 10 was applied for the use of a LOAEL, a UF_A of 10 was applied to account for interspecies toxicokinetic and toxicodynamic differences, and a UF of 10 was applied to account for differences in sensitive populations, for a total UF of 1,000.
(c) A UF_L of 10 was applied for the use of a LOAEL, a UF_A of 3 was applied to account for interspecies toxicodynamic uncertainty, and a UF_H of 3 was applied to account for differences in sensitive populations, for a total UF of 100.

Table 5.2 US EPA TCE Non-cancer Chronic Inhalation Toxicity Values

Chemical	POD (mg/m^3 [ppm])	UF	Candidate RfC ($\mu g/m^3$ [ppb])	RfC ($\mu g/m^3$ [ppb])	Effect/Source
TCE	0.021 (0.0037)	10[a]	2.1 (0.37)	2 (0.4)	Increased fetal cardiac malformations in rats (Johnson *et al.*, 2003)
	0.19 (0.033)	100[b]	1.9 (0.33)		Decreased thymus weight in adult female mice (Keil *et al.*, 2009)

Notes:
$\mu g/m^3$ = Microgram per Cubic Meter; LOAEL = Lowest Observed Adverse Effect Level; mg/m^3 = Milligram per Cubic Meter; POD = Point of Departure; ppb = Parts per Billion; ppm = Parts per Million; RfC = Reference Concentration; TCE = Trichloroethylene; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: US EPA (2011a,b).
(a) A UF_A of 3 was applied to account for interspecies toxicodynamic differences, and a UF of 3 was applied to account for differences in sensitive populations, for a total UF of 10.
(b) A UF_L of 10 was applied for the use of a LOAEL, a UF_A of 3 was applied to account for interspecies toxicodynamic uncertainty, and a UF_H of 3 was applied to account for differences in sensitive populations, for a total UF of 100.

5.2.1.2 ATSDR Toxicity Criteria

I reviewed ATSDR's toxicological profile for TCE (ATSDR, 2019a) and its PHA for Camp Lejeune drinking water (ATSDR, 2017a) to identify the current ATSDR MRLs for TCE and the TCE toxicity values applied in the PHA for evaluation of human health risk. In its 2019 toxicological profile for TCE, and in the PHA (ATSDR, 2017a), ATSDR adopted the US EPA chronic RfD and values for TCE (described above) as the MRLs for oral and inhalation exposures, respectively, for both chronic and subchronic (intermediate) exposure durations.

In addition to the RfD, RfC, and MRL values for TCE, the ATSDR PHA also derived specific target organ toxicity doses (TTDs) for several endpoints, including neurological effects, *via* oral and inhalation routes of exposure (ATSDR, 2017a). The oral and inhalation TTDs for neurological effects are summarized in Table 5.3. Although the PHA (ATSDR, 2017a) provides information on the effect, it does not specifically describe the derivation of the TTDs, nor does it report the underlying studies that provide the basis of the values. However, based on my review of the ATSDR toxicological profile for TCE (2019a) and US EPA's toxicological review for TCE (US EPA, 2011a), it is likely that the ATSDR PHA relied on the studies of Gash *et al*. (2008) and Arito *et al*. (1994) to determine TTDs for neurological effects of TCE *via* oral and inhalation routes of exposure (Table 5.3).

Table 5.3 ATSDR TCE TTDs for Neurological Effects *via* the Oral and Inhalation Routes of Exposure

Chemical	POD (mg/kg-day)	POD (µg/m³)	UF	TTD_{neuro}	Effect / *Likely* Source
TCE	1,000	–	1,000[a]	1 mg/kg-day	Subchronic oral study observed decreased dopaminergic neurons in rats (Gash *et al.*, 2008)
	–	63,930[b]	1,000[b]	64 µg/m³ (11.9 ppb)	Subchronic oral study observed decreased wakefulness in rats (Arito *et al.*, 1994)[b]

Notes:
µg/m³ = Microgram per Cubic Meter; ATSDR = Agency for Toxic Substances and Disease Registry; LOAEL = Lowest Observed Adverse Effect Level; mg/kg-day = Milligram per Kilogram Body Weight per Day; mg/m³ = Milligram per Cubic Meter; PHA = Public Health Assessment; POD = Point of Departure; ppb = Parts per Billion; ppm = Parts per Million; TCE = Trichloroethylene; TTD_{neuro} = Target Organ Toxicity Dose for Neurological Effects; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; UF_{Schr} = Subchronic to Chronic Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: ATSDR (2017a).
(a) Although not discussed in the ATSDR PHA (ATSDR, 2017a), ATSDR (2019a) provides a LOAEL for Gash *et al.* (2008) of 1,000 mg/kg-day. The UFs are not discussed in either document.
(b) Although not discussed in the ATSDR PHA (ATSDR, 2017a), ATSDR (2019a) and US EPA (2011a) provide a LOAEL for Arito *et al.* (1994) of 50 ppm, which is equivalent to 269 mg/m³ (1 ppm TCE = 5.37 mg/m³ TCE). Based on an exposure of 8 hours/day, 5 days/week, for 6 weeks, US EPA (2011a) calculated a continuous human equivalent concentration of ~64,000 µg/m³. The composite UF of 1,000 described by US EPA (2011a) is as follows: UF_{Schr} = 3 for subchronic to chronic uncertainty, UF_A = 3 for animal to human uncertainty, UF_H = 10 for human variability, and UF_L = 10 for use of a LOAEL.

For the toxicity value of 64 µg/m³, also described by US EPA in its toxicological review for TCE, US EPA (2011a) included a UF of 3 to adjust from a subchronic study to a chronic exposure duration. Therefore, I have removed that UF to adjust the value to reflect a subchronic exposure duration (64 µg/m³ × 3), resulting in subchronic TCE toxicity criterion of 192 µg/m³ (0.036 parts per million [ppm]) that can be applied for subchronic exposure durations (*i.e.*, less than 7 years of exposure per US EPA guidelines) for neurological effects.

5.2.1.3 TCE Toxicity Criteria Applied in the Risk Calculations

Because US EPA's and ATSDR's toxicity values for TCE (that are based on the most sensitive endpoints) are not based on neurological effects, I applied ATSDR's neurological TTDs for TCE risk calculations in this report. It is notable that although the endpoints are neurological, they are not necessarily related to PD (*e.g.*, the inhalation value is based on wakefulness). However, because toxicity criteria are derived to be protective of the most sensitive endpoint, the neurological effect toxicity criteria are considered protective of all neurological effects evaluated for TCE, including effects related to PD. The toxicity criteria used in the TCE risk calculations are summarized in Table 5.4.

As discussed in Section 3, exposures less than 7 years are considered subchronic. Thus, subchronic non-cancer toxicity criteria are applied when exposure durations are less than 7 years.

Table 5.4 TCE Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Duration	Endpoint	Value
TCE	Oral TTD	Chronic/Subchronic	Neurological	1 mg/kg-day
	Inhalation TTD	Chronic	Neurological	64 µg/m³ (11.9 ppb)
	Inhalation TTD	Subchronic	Neurological	192 µg/m³ (36 ppb)

Notes:
µg/m³ = Microgram per Cubic Meter; mg/kg-day = Milligram per Kilogram Body Weight per Day; ppb = Parts per Billion; TCE = Trichloroethylene; TTD= Target Organ Toxicity Dose.

5.2.2 Tetrachloroethylene (PCE)

5.2.2.1 US EPA Toxicity Criteria (RfDs and RfCs)

Table 5.5 summarizes the PODs, total UFs, candidate RfDs and effects associated with each, and the final RfD US EPA derived for PCE (US EPA, 2012b). Table 5.6 summarizes the PODs, UFs, candidate RfCs and effects associated with each, and the final RfC US EPA derived for PCE (US EPA, 2012b).

Based on the hazard assessment for PCE, US EPA first derived two candidate RfCs from two human PCE studies (US EPA, 2012b); one based on reaction time and cognitive effects from worker inhalation exposures (Echeverria *et al.*, 1995), and one based on color vision changes from worker inhalation exposures (Cavalleri *et al.*, 1994) (Table 5.6). US EPA then applied a PCE PBPK model to conduct a route-to-route (inhalation-to-oral) extrapolation to derive the PCE RfDs from the same studies used for the PCE RfC derivations (Table 5.5).

Table 5.5 US EPA PCE Non-cancer Chronic Oral Toxicity Values

Chemical	POD (mg/kg-day)	UF	Candidate RfD (mg/kg-day)	RfD (mg/kg-day)	Effect/Source
PCE	9.7	1,000[a]	0.0097	0.006	Reaction time, cognitive effects in occupationally exposed adults (Echeverria *et al.*, 1995)
	2.6	1,000[a]	0.0026		Color vision changes in occupationally exposed adults (Cavalleri *et al.*, 1994)

Notes:
LOAEL = Lowest Observed Adverse Effect Level; mg/kg-day = Milligram per Kilogram Body Weight per Day; NOAEL = No Observed Adverse Effect Level; PCE = Tetrachloroethylene; POD = Point of Departure; RfD = Reference Dose; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: US EPA (2012b).
(a) A total UF of 1,000 was used for both candidate RfDs and comprised a UF_H of 10 for human variability, a UF_L of 10 for extrapolation from a LOAEL to a NOAEL, and a UF_D of 10 for database uncertainty.

Table 5.6 US EPA PCE Non-cancer Chronic Inhalation Toxicity Values

Chemical	POD (mg/m^3)	UF	Candidate RfC ($\mu g/m^3$ [ppb])	RfC ($\mu g/m^3$ [ppb])	Effect/Source
PCE	56	1,000[a]	56 (8)	40 (6)	Reaction time, cognitive effects in occupationally exposed adults (Echeverria *et al.*, 1995)
	15	1,000[a]	15 (2)		Color vision changes in occupationally exposed adults (Cavalleri *et al.*, 1994)

Notes:
$\mu g/m^3$ = Microgram per Cubic Meter; LOAEL = Lowest Observed Adverse Effect Level; mg/m^3 = Milligram per Cubic Meter; NOAEL = No Observed Adverse Effect Level; PCE = Tetrachloroethylene; POD = Point of Departure; ppb = Parts per Billion; RfC = Reference Concentration; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: US EPA (2012b).
(a) A total UF of 1,000 was used for both candidate RfCs and comprised a UF_H of 10 for human variability, a UF_L of 10 for extrapolation from a LOAEL to a NOAEL, and a UF_D of 10 for database uncertainty.

5.2.2.2 ATSDR Toxicity Criteria

I reviewed ATSDR's toxicological profile for PCE (ATSDR, 2019b) and its PHA for Camp Lejeune drinking water (ATSDR, 2017a) to identify the current ATSDR MRLs for PCE and the PCE toxicity values applied in the PHA for evaluation of human health risk. In its 2019 toxicological profile for PCE, and in the PHA (ATSDR, 2017a), ATSDR derived 0.008 mg/kg-day and 0.04 mg/m^3 (0.006 ppm) values for PCE as the MRLs for oral and inhalation exposures, respectively, for both chronic and subchronic (intermediate) exposure durations, based on the same study that US EPA used (Cavelleri *et al*., 1994). The oral MRL is similar to US EPA's RfD and the inhalation MRL is the same as US EPA's RfC. Table 5.7 summarizes the PCE oral MRL.

Table 5.7 ATSDR PCE Non-cancer Oral Toxicity Values

Chemical	Exposure Duration	POD (mg/kg-day)	Combined UF	MRL (mg/kg-day)	Effect/Source
PCE	Intermediate and Chronic	2.3	300[a]	0.008	Color vision changes in occupationally exposed adults (Cavalleri *et al*., 1994)

Notes:
ATSDR = Agency for Toxic Substances and Disease Registry; LOAEL = Lowest Observed Adverse Effect Level; mg/kg-day = Milligram per Kilogram Body Weight per Day; NOAEL = No Observed Adverse Effect Level; PCE = Tetrachloroethylene; POD = Point of Departure; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor.
Source: ATSDR (2019b).
(a) ATSDR (2019b) applied a UF_L of 10 for extrapolation from a LOAEL to a NOAEL, a UF_H of 10 for human variability, and a UF_D of 3 for database uncertainty.

Note that ATSDR reports neurological effect TTDs for PCE in its PHA for Camp Lejeune (ATSDR, 2017a) that are equal to the US EPA RfD and RfC for PCE.

5.2.2.3 PCE Toxicity Criteria Applied in the Risk Calculations

The toxicity criteria used in the PCE risk calculations are summarized in Table 5.8. It is notable that although the endpoints are neurological, they are not necessarily related to PD (*e.g*., one of the oral values is based on color vision changes). However, because the endpoints are based on the most sensitive endpoints from the most reliable animal and human studies, they are considered protective of other health effects, including PD.

Table 5.8 PCE Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Duration	Endpoint	Value
PCE	Oral RfD	Chronic/Subchronic	Neurological	0.006 mg/kg-day
	Inhalation RfC	Chronic/Subchronic	Neurological	40 µg/m^3 (6 ppb)

Notes:
µg/m^3 = Microgram per Cubic Meter; mg/kg-day = Milligram per Kilogram Body Weight per Day; PCE = Tetrachloroethylene; ppb = Parts per Billion; RfC = Reference Concentration; RfD = Reference Dose.

5.2.3 Benzene

5.2.3.1 US EPA Toxicity Criteria (RfDs and RfCs)

Table 5.9 summarizes the POD, total UF, associated health effects, and the final RfD US EPA derived for benzene (US EPA, 2002b). Table 5.10 summarizes the POD, total UF, and the final RfC US EPA derived for benzene (US EPA, 2002b). Based on the hazard assessment for benzene, US EPA (2002b) derived an RfC for benzene based on decreased absolute lymphocyte count (ALC) in workers following chronic inhalation exposure to benzene (Rothman *et al.*, 1996) (Table 5.10). US EPA then conducted an inhalation-to-oral extrapolation to derive a benzene RfD from the same study used to derive the benzene RfC (Table 5.9).

Table 5.9 US EPA Benzene Non-cancer Chronic Oral Toxicity Value

Chemical	POD (mg/kg-day)	UF	RfD (mg/kg-day)	Effect/Source
Benzene	1.2	300[a]	0.004	Decreased absolute lymphocyte count (ALC) in a human occupational study (Rothman *et al.*, 1996)

Notes:
LOAEL = Lowest Observed Adverse Effect Level; mg/kg-day = Milligram per Kilogram Body Weight per Day; NOAEL = No Observed Adverse Effect Level; POD = Point of Departure; RfD = Reference Dose; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; UF_{Schr} = Subchronic to Chronic Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: US EPA (2002b).
(a) US EPA (2002b) applied a UF_L of 3 for extrapolation from a LOAEL to NOAEL, a UF_H of 10 for human variability, a UF_{Schr} of 3 for subchronic to chronic exposure, and a UF_D of 3 for database uncertainty.

Table 5.10 US EPA Benzene Non-cancer Chronic Inhalation Toxicity Value

Chemical	POD (µg/m³ [ppb])	UF	RfC (µg/m³ [ppb])	Effect/Source
Benzene	8,200 (2,600)	300[a]	30 (9)	Decreased absolute lymphocyte (ALC) count in a human occupational study (Rothman *et al.*, 1996)

Notes:
µg/m³ = Microgram per Cubic Meter; LOAEL = Lowest Observed Adverse Effect Level; NOAEL = No Observed Adverse Effect Level; POD = Point of Departure; ppb = Parts per Billion; RfC = Reference Concentration; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; UF_{Schr} = Subchronic to Chronic Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: US EPA (2002b).
(a) US EPA applied a UF_L of 3 for extrapolation from a LOAEL to NOAEL, a UF_H of 10 for human variability, a UF_{Schr} of 3 for subchronic to chronic exposure, and a UF_D of 3 for database uncertainty.

5.2.3.2 ATSDR Toxicity Criteria

I reviewed ATSDR's toxicological profile for benzene (ATSDR, 2007a) and its PHA for Camp Lejeune drinking water (ATSDR, 2017a) to identify the current ATSDR MRLs for benzene and the benzene toxicity values applied in the PHA for evaluation of human health risk. Tables 5.11 and 5.12 summarize the PODs, total UFs, and MRLs derive by ATSDR for benzene.

ATSDR (2007a) derived a chronic oral MRL for benzene based on the chronic occupational inhalation study conducted by Lan *et al*. (2004) (see discussion below) and conducting an inhalation-to-oral extrapolation (Table 5.11). ATSDR (2007a) derived an intermediate inhalation MRL for benzene based on delayed immune response effects in mice following inhalation exposure to benzene (Rosenthal and Snyder, 1987), and derived a chronic inhalation MRL for benzene based on decreased B-cell counts in a study of occupationally exposed workers (Lan *et al*., 2004) (Table 5.12).

Table 5.11 ATSDR Benzene Non-cancer Oral Toxicity Value

Chemical	Exposure Duration	POD (mg/kg-day)	UF	MRL (mg/kg-day)	Effect/Source
Benzene	Chronic	0.014	30[a]	0.0005	B-cell count in occupationally exposed workers (Lan *et al*., 2004)

Notes:
ATSDR = Agency for Toxic Substances and Disease Registry; mg/kg-day = Milligram per Kilogram Body Weight per Day; MRL = Minimal Risk Level; POD = Point of Departure; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor.
Source: ATSDR (2007a).
(a) ATSDR (2007a) applied a UF_H of 10 for human variability and a UF_D of 3 for the uncertainty in route-to-route extrapolation, for a total UF of 30.

Table 5.12 ATSDR Benzene Non-cancer Inhalation Toxicity Values

Chemical	Exposure Duration	POD (mg/m^3 [ppb])	UF	MRL ($\mu g/m^3$ [ppb])	Effect/Source
Benzene	Intermediate	5.8 (1.8)	300[a]	20 (6)	Immune effects in mice (Rosenthal and Snyder, 1987)
	Chronic	0.096 (0.03)	10[b]	9.6 (3)	B-cell counts in occupationally exposed workers (Lan *et al*., 2004)

Notes:
$\mu g/m^3$ = Microgram per Cubic Meter; ATSDR = Agency for Toxic Substances and Disease Registry; LOAEL = Lowest Observed Adverse Effect Level; mg/m^3 = Milligram per Cubic Meter; MRL = Minimal Risk Level; NOAEL = No Observed Adverse Effect Level; POD = Point of Departure; ppb = Parts per Billion; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor.
Source: ATSDR (2007a).
(a) ATSDR (2007a) applied a UF_L of 10 for extrapolation from a LOAEL to a NOAEL, a UF_A of 3 for extrapolation from animals to humans, and a UF_H of 10 for human variability.
(b) ATSDR (2007a) applied a UF_H of 10 for human variability.

In addition to the RfD, RfC, and MRL values for benzene, in its PHA for Camp Lejeune drinking water, ATSDR derived a TTD for neurological effects *via* the oral route of exposure (ATSDR, 2017a). The POD, total UF, and oral TTD for neurological effects for benzene is summarized in Table 5.13. The PHA (ATSDR, 2017a), however, does not specifically describe the derivation of the TTD, nor does it report the underlying study that provides the basis for this value. However, the POD is generally consistent with the lowest intermediate exposure NOAEL (no observed adverse effect level) (8 mg/kg-day) and LOAEL (lowest adverse effect level) (8 mg/kg-day) for neurological effects reported in mice in the benzene toxicological profile (ATSDR, 2007a, Table 3-2). ATSDR did not derive an inhalation TTD for neurological effects for benzene.

Table 5.13 ATSDR Benzene TTD for Neurological Effects *via* the Oral Route of Exposure

Chemical	POD (mg/kg-day)	UF	TTD_{neuro} (mg/kg-day)	Effect	Source
Benzene	15	100[a]	0.15	Not provided	Not provided

Notes:
ATSDR = Agency for Toxic Substances and Disease Registry; mg/kg-day = Milligram per Kilogram Body Weight per Day; POD = Point of Departure; TTD_{neuro} = Target Organ Toxicity Dose for Neurological Effects; UF = Uncertainty Factor.
Source: ATSDR (2017a).
(a) ATSDR (2017a) did not discuss the basis of the UFs.

5.2.3.3 Benzene Toxicity Criteria Applied in the Risk Calculations for the Plaintiff

Because US EPA's and ATSDR's toxicity values for benzene (that are based on the most sensitive endpoints) are not based on neurological effects, I applied ATSDR's neurological TTD for benzene risk calculations in this report (*i.e.*, for the oral route of exposure only, because an inhalation value was not provided). Although the endpoint is reported to be neurological, based on the discussion in Section 5.1.3, it is unlikely to be related to PD. However, the value should be considered protective of other neurological health effects, including PD. The toxicity criteria used in the benzene non-cancer risk calculations are summarized in Table 5.14.

Table 5.14 Benzene Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Duration	Endpoint	Value
Benzene	Oral TTD	Chronic/Subchronic	Neurological	0.15 mg/kg-day
	Inhalation MRL	Chronic	B-cell counts	9.6 µg/m³
	Inhalation MRL	Subchronic	Immune effects	20 µg/m³

Notes:
µg/m³ = Microgram per Cubic Meter; mg/kg-day = Milligram per Kilogram Body Weight per Day; MRL = Minimal Risk Level; TTD= Target Organ Toxicity Dose.

As discussed in Section 3, exposures less than 7 years are considered subchronic. Thus, subchronic non-cancer toxicity criteria are applied when exposure durations are less than 7 years.

5.2.4 Vinyl Chloride

5.2.4.1 US EPA Toxicity Criteria (RfDs and RfCs)

Table 5.15 summarizes the PODs, total UF, associated health effects, and the final RfD US EPA derived for vinyl chloride (US EPA, 2003). Table 5.16 summarizes the PODs, total UFs, associated health effects, and the final RfC US EPA derived for vinyl chloride (US EPA, 2003).

Based on the hazard assessment for vinyl chloride, US EPA (2003) derived an RfD for vinyl chloride based on liver polymorphism effects in rats following chronic exposure to vinyl chloride in the diet (Til *et al.*, 1982; US EPA, 2003) (Table 5.15). US EPA then applied a vinyl chloride PBPK model to conduct a route-to-route (oral-to-inhalation) extrapolation to derive the vinyl chloride RfC from the same study used for the vinyl chloride RfC derivation (US EPA, 2003) (Table 5.16).

Table 5.15 US EPA Vinyl Chloride Non-cancer Chronic Oral Toxicity Value

Chemical	POD (mg/kg-day)	UF	RfD (mg/kg-day)	Effect/Source
Vinyl chloride	0.09	30[a]	0.003	Liver cell polymorphism in rat chronic feed study (Til *et al.*, 1982; US EPA, 2003)

Notes:
mg/kg-day = Milligram per Kilogram Body Weight per Day; POD = Point of Departure; RfD = Reference Dose; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: US EPA (2003).
(a) A UF_H of 10 was applied for human variability and a UF_A of 3 was applied for animal-to-human extrapolation to account for toxicodynamic differences between species.

Table 5.16 US EPA Vinyl Chloride Non-cancer Chronic Inhalation Toxicity Value

Chemical	POD (µg/m³ [ppb])	UF	RfC (µg/m³ [ppb])	Effect/Source
Vinyl chloride	2,500 (~100)	30[a]	100 (~39)	Liver cell polymorphism in rat chronic feed study (Til *et al.*, 1982; US EPA, 2003)

Notes:
µg/m³ = Microgram per Cubic Meter; POD = Point of Departure; ppb = Parts per Billion; RfC = Reference Concentration; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: US EPA (2003).
(a) A UF_H of 10 was applied for human variability and a UF_A of 3 was applied for animal-to-human extrapolation to account for toxicodynamic differences between species.

5.2.4.2 ATSDR Toxicity Criteria

I reviewed ATSDR's toxicological profile for vinyl chloride (ATSDR, 2024b) and its PHA for Camp Lejeune drinking water (ATSDR, 2017a) to identify the current ATSDR MRLs for vinyl chloride and the vinyl chloride toxicity values applied in the PHA for evaluation of human health risk. In its 2024 toxicological profile for vinyl chloride, ATSDR (2024b) concluded that there were insufficient data for vinyl chloride for the derivation of an intermediate-duration oral MRL or for derivation of a chronic-duration inhalation MRL for vinyl chloride. ATSDR (2024b) derived a chronic-duration oral MRL for vinyl chloride, equal to the US EPA RfD, and derived the same way as the US EPA RfD, from the same studies (see above).

ATSDR (2024b) also derived an intermediate-duration inhalation MRL for vinyl chloride based on increased incidence of centrilobular hypertrophy of the liver in female rat offspring following inhalation exposure to vinyl chloride during gestation and lactation (Thornton *et al.*, 2002). The MRL derivation is summarized in Table 5.17.

Table 5.17 ATSDR Vinyl Chloride Non-cancer Inhalation Toxicity Value

Chemical	Exposure Duration	POD (µg/m³ [ppb])	UF	MRL (µg/m³ [ppb])	Effect/Source
Vinyl chloride	Intermediate	1,500 (512.5)	30[a]	50 (20)	Increased incidence of centrilobular hypertrophy of the liver in rats (Thornton *et al.*, 2002)

Notes:
µg/m³ = Microgram per Cubic Meter; ATSDR = Agency for Toxic Substances and Disease Registry; MRL = Minimal Risk Level; POD = Point of Departure; ppb = Parts per Billion; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor.
Source: ATSDR (2024b).
(a) A UF_H of 10 was applied for human variability and a UF_A of 3 was applied for animal-to-human extrapolation to account for toxicodynamic differences between species.

ATSDR did not derive neurological TTDs for vinyl chloride in its PHA for Camp Lejeune (ATSDR, 2017a).

5.2.4.3 Vinyl Chloride Toxicity Criteria Applied in the Risk Calculations

The toxicity criteria used in the vinyl chloride non-cancer risk calculations (protective of the most sensitive endpoints, including neurological effects) are summarized in Table 5.18.

Table 5.18 Vinyl Chloride Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Duration	Endpoint	Value
Vinyl chloride	Oral RfD	Chronic/Subchronic	Liver polymorphism	0.003 mg/kg-day
	Inhalation MRL	Chronic/Subchronic	Centrilobular hypertrophy of the liver	50 µg/m³

Notes:
µg/m³ = Microgram per Cubic Meter; mg/kg-day = Milligram per Kilogram Body Weight per Day; MRL = Minimal Risk Level; RfD = Reference Dose.

5.2.5 *trans*-1,2-Dichloroethylene (1,2-tDCE)

5.2.5.1 US EPA Toxicity Criteria (RfDs and RfCs)

Table 5.19 summarizes the POD, total UF, associated health effects, and the final RfD US EPA derived for 1,2-tDCE (US EPA, 2010a,b). Table 5.20 summarizes the PODs, total UFs, associated health effects, and the final RfC US EPA derived for 1,2-tDCE (US EPA, 2010a,b).

Based on the hazard assessment for 1,2-tDCE, US EPA (2010a,b) derived an RfD for 1,2-tDCE based on a decreased number of antibody-forming cells (AFCs) against sheep red blood cells (sRBCs) in male mice following exposure to 1,2-tDCE in drinking water (Shopp *et al.*, 1985) (Table 5.19). US EPA (2020c) also derived subchronic and chronic provisional reference concentration (p-RfC) values for 1,2-tDCE based on a subchronic inhalation toxicity study in which decreased lymphocyte counts was observed in male rats (Kelly, 1998) (Table 5.20).

Table 5.19 US EPA 1,2-tDCE Non-cancer Chronic Oral Toxicity Value

Chemical	POD (mg/kg-day)	UF	RfD (mg/kg-day)	Effect/Source
1,2-tDCE	65	3,000[a]	0.02	Immune effects (Shopp *et al.*, 1985)

Notes:
1,2-tDCE = *trans*-1,2-Dichloroethylene; mg/kg-day = Milligram per Kilogram Body Weight per Day; POD = Point of Departure; RfD = Reference Dose; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_{Schr} = Subchronic to Chronic Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Sources: US EPA (2010a,b).
(a) A UF_H of 10 was applied for human variability, a UF_A of 10 was applied for animal-to-human extrapolation, a UF_D of 3 was applied for database deficiencies, and a UF_{Schr} of 10 was applied for extrapolation from subchronic to chronic exposure.

Table 5.20 US EPA 1,2-tDCE Non-cancer Provisional Subchronic and Provisional Chronic Inhalation Toxicity Values

Chemical	Exposure Duration	POD mg/m^3 (ppm)	UF	p-RfC $\mu g/m^3$ (ppb)	Effect/Source
1,2-tDCE	Subchronic	109 (27.5)	300[a]	400 (100)	Immune effects – Decreased lymphocyte counts in male mice (Kelly, 1998)
	Chronic	109 (27.5)	3,000[b]	40 (10)	

Notes:
1,2-tDCE = *trans*-1,2-Dichloroethylene; $\mu g/m^3$ = Microgram per Cubic Meter; mg/m^3 = Milligram per Cubic Meter; POD = Point of Departure; ppb = Parts per Billion; ppm = Parts per Million; p-RfC = Provisional Reference Concentration; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_{Schr} = Subchronic to Chronic Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: US EPA (2020c).
(a) A UF_H of 10 was applied for human variability, a UF_A of 3 was applied for animal-to-human extrapolation to account for toxicodynamic differences between species, and a UF_D of 10 was used to account for database uncertainty.
(b) The same UFs listed in note (a) were applied, plus an additional UF_{Schr} of 10 to account for the use of a subchronic study.

5.2.5.2 ATSDR Toxicity Criteria

I reviewed ATSDR's toxicological profile for 1,2-tDCE (ATSDR, 2023) and its PHA for Camp Lejeune (ATSDR, 2017a) to identify the current ATSDR MRLs for 1,2-tDCE and the 1,2-tDCE toxicity values applied in the PHA for evaluation of human health risk. In its 2023 toxicological profile for 1,2-tDCE, using the same study (Shopp *et al.*, 1985) that US EPA used to derive an oral RfD for 1,2-tDCE, ATSDR (2023) derived a provisional intermediate duration oral MRL for 1,2-tDCE. The intermediate MRL derivation is summarized in Table 5.21. ATSDR (2023) concluded that there was insufficient data for 1,2-tDCE for the derivation of a chronic-duration oral MRL.

Table 5.21 ATSDR 1,2-tDCE Non-cancer Oral Toxicity Value

Chemical	Exposure Duration	POD (mg/kg-day)	UF	MRL (mg/kg-day)	Effect/Source
1,2-tDCE	Intermediate	16.75	100[a]	0.2	Decreased humoral immunity (Shopp *et al.*, 1985)
	Chronic	–	–	–	–

Notes:
1,2-tDCE = *trans*-1,2-Dichloroethylene; ATSDR = Agency for Toxic Substances and Disease Registry; mg/kg-day = Milligram per Kilogram Body Weight per Day; MRL = Minimal Risk Level; POD = Point of Departure; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor.
Source: ATSDR (2023).
(a) A UF_A of 10 was applied for animal-to-human extrapolation, and a UF_H of 10 was applied for human variability.

In addition to the RfD, RfC, and MRL values for 1,2-tDCE, in its PHA for Camp Lejeune, ATSDR derived a TTD for neurological effects *via* the oral route of exposure (ATSDR, 2017a). The oral TTD for neurological effects for 1,2-tDCE is summarized in Table 5.22. Although the PHA (ATSDR, 2017a) provides information on the effect, it does not specifically describe the derivation of the TTD, nor does it report the underlying study that provides the basis of the value. However, the POD of 336 mg/kg-day is approximately 10-fold lower than the lowest intermediate exposure NOAEL for neurological effects (3,245 mg/kg-day in rats) described in the 1,2-tDCE toxicological profile (ATSDR, 2023, Table 2-2). Application of a UF_A of 10 for animal to human extrapolation, and a UF_H of 10 for human variability, for a total UF of 100, would result in a TTD of 32 mg/kg-day if derived from the NOAEL of 3,245 mg/kg-day. Therefore, the value derived by ATSDR in the PHA (3.36 mg/kg-day) is about 10-fold more protective than what the neurological studies in the toxicological profile suggest (ATSDR, 2023).

An inhalation neurological TTD was not derived for 1,2-tDCE in ATSDR's PHA for Camp Lejeune (ATSDR, 2017a).

Table 5.22 ATSDR 1,2-tDCE TTD for Neurological Effects *via* the Oral Route of Exposure

Chemical	POD (mg/kg-day)	UF	TTD_{neuro} (mg/kg-day)	Effect	Source
1,2-tDCE	336	100[a]	3.36	Acute ataxia	Not provided

Notes:
1,2-tDCE = trans-1,2-Dichloroethylene; ATSDR = Agency for Toxic Substances and Disease Registry; mg/kg-day = Milligram per Kilogram Body Weight per Day; POD = Point of Departure; TTD_{neuro} = Target Organ Toxicity Dose for Neurological Effects; UF = Uncertainty Factor.
Source: ATSDR (2017a).
(a) UFs not discussed by ATSDR.

5.2.5.3 1,2-tDCE Toxicity Criteria Applied in the Risk Calculations for the Plaintiff

The toxicity criteria used in the 1,2-tDCE non-cancer risk calculations (protective of the most sensitive endpoints, including neurological effects) are summarized in Table 5.23.

Table 5.23 1,2-tDCE Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Duration	Endpoint	Value
1,2-tDCE	Oral TTD	Chronic/Subchronic	Neurological	3.36 mg/kg-day
	Inhalation RfC	Chronic	Immunological	40 µg/m³
	Inhalation RfC	Subchronic	Immunological	400 µg/m³

Notes:
1,2-tDCE = *trans*-1,2-Dichloroethylene; µg/m³ = Microgram per Cubic Meter; mg/kg-day = Milligram per Kilogram Body Weight per Day; RfC = Reference Concentration; TTD = Target Organ Toxicity Dose.

As discussed in Section 3, exposures less than 7 years are considered subchronic. Thus, subchronic non-cancer toxicity criteria are applied when exposure durations are less than 7 years.

9.2 Dr. Schwarz

Dr. Schwarz's report (Schwarz, 2025) does not provide a reliable analysis of specific causation or risk of PD with regard to Mr. Sparks' alleged exposures. Dr. Schwarz (2025) concludes that Mr. Sparks' PD is "more likely than not due to his exposure to TCE at Camp Lejeune" without providing a robust analysis of the best available scientific information relevant to the potential causal association between exposure to these chemicals and PD. Below, I describe several flaws in Dr. Schwarz's analysis:

- Dr. Schwarz's risk evaluation is not consistent with US EPA's risk assessment guidelines, which consider not only exposure concentrations, but also exposure frequency and duration.
    - As discussed in Sections 3 and 5, exposure frequency and duration are critical components of US EPA's risk assessment methodology. It is only when the exposure concentrations, in combination with exposure frequencies and durations, result in doses exceeding US EPA's toxicity criteria (*i.e.*, result in a risk estimate that exceeds US EPA's acceptable targets) that there is concern for potential adverse health effects. And even with slight exceedances of US EPA's conservative risk targets, health effects are not necessarily expected (discussed in Section 3).
- Dr Schwarz (2025) relies on Dr. Reynolds' exposure charts as support that Mr. Sparks' exposures were "substantial," but provides no basis for this conclusion other than simply pointing to the total mass values reported by Dr. Reynolds (2025a). As discussed in the previous section, estimates of total chemical mass exposure over time cannot be used directly to evaluate potential health effects for the plaintiff, because there are no total mass exposure estimates from relevant animal or epidemiology studies against which to make reliable risk-based comparisons. Exposures need to be estimated as oral doses of mg/kg-day or inhalation concentrations of $\mu g/m^3$, per US EPA risk assessment guidelines. Adding up mass over many days and months will, undoubtedly, result in a very large value, but it is an incorrect value for the purpose of risk evaluation. Therefore, Dr. Schwarz's conclusions based on estimates of total chemical mass exposure for Mr. Sparks are meaningless, misleading, and cannot be relied upon for risk evaluation for Mr. Sparks.
- Dr. Schwarz's comparison to US EPA maximum contaminant levels (MCLs) for allowable chemical concentrations in drinking water is not a reliable risk evaluation method.
    - US EPA does not use MCLs to evaluate potential risks to human health.
    - MCLs are derived to be acceptable (health-protective) daily drinking water concentrations over a lifetime of exposure (~70 years) (US EPA, 2024), which is much longer than Mr. Sparks' approximately 1.25 years of exposure during his time at Camp Lejeune.
    - Further, the MCLs for TCE, PCE, vinyl chloride, and benzene are based on cancer health effects and not PD; therefore, an MCL exceedance for these chemicals, even over a longer period of time than the plaintiff was exposed, is not relevant to PD.
    - Therefore, a simple comparison of drinking water concentrations to MCLs, without considering exposure duration or the health effect on which the MCL is based, is not consistent with standard risk assessment practice, and is misleading.

- Dr. Schwarz's report refers to exposure information from several Camp Lejeune studies to support her conclusions. However, as discussed in Dr. Goodman's report (Goodman, 2025), there are methodological limitations in these studies (*e.g.*, high likelihood of exposure misclassification). In addition, with regard to the Camp Lejeune studies, overall, Dr. Goodman states the following:

> Overall, there were no consistent associations reported between either working or living at Camp Lejeune or TCE, PCE, benzene, or vinyl chloride exposures at Camp Lejeune and PD. Most risk estimates were small and statistically null, and the few statistically significant risk estimates had wide CIs and were not reported across other analyses of the Camp Lejeune population, indicating a high likelihood of bias or confounding, such that they do not provide evidence of a causal link between exposure to contaminated water at Camp Lejeune and PD. (Goodman, 2025).

- In addition, as discussed in Section 5, based on a comprehensive review of the best available and most current epidemiology and animal studies, Dr. Goodman (2025) concludes that the scientific evidence does not support a causal association between TCE, PCE, benzene, vinyl chloride, or 1,2-tDCE exposure and PD.

As discussed in my report (Section 6), applying standard risk assessment methodology (*i.e.*, considering exposure concentrations in addition to exposure frequency and duration for the plaintiff), the hazard indices (HIs) estimated for Mr. Sparks' exposures, for neurological effects for a healthy worker population, do not exceed US EPA's acceptable HI target.

Therefore, Dr. Reynolds' and Dr. Schwarz's expert reports do not change my opinions, as discussed in my report and summarized in Section 10, regarding Mr. Sparks' claim that exposures from Camp Lejeune are the cause of his PD.